

FEB 15 2007

Application No.: 10/627,372

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Docket No.: 421842000400

**BEST AVAILABLE COPY****AMENDMENTS TO THE CLAIMS**

The following list of claims replaces all prior versions and lists of claims:

Claim 1 (Currently amended): A method for ~~inhibiting~~ decreasing an incidence of a new onset of type 2 diabetes mellitus, or treating an inflammatory or metabolic disorder selected from the group consisting of type 2 diabetes mellitus, metabolic syndrome, and inflammation caused by osteoarthritis in a mammal by administering to the mammal in need thereof, a therapeutically effective amount of telmisartan, or an analog thereof, sufficient to (a) at least partially activate peroxisome proliferator activated receptors (PPARs) and (b) at least partially inhibit, antagonize or block an activity of angiotensin II type 1 receptors.

Claim 2 (Currently amended): The method of claim 1 wherein the ~~inhibiting~~ decreasing the incidence of the new onset of type 2 diabetes mellitus, or treating the inflammatory or metabolic disorder does not cause, promote, or aggravate fluid retention, peripheral edema, pulmonary edema, or congestive heart failure in the mammal.

Claim 3 (Previously presented): The method of claim 1 wherein the telmisartan, or an analog thereof, is administered in a pharmaceutically acceptable form.

Claim 4 (Previously presented): The method of claim 1 wherein said telmisartan, or an analog thereof, is administered in a therapeutically effective amount sufficient to inhibit, slow, or delay development of at least one metabolic disorder or disease selected from the group consisting of insulin resistance, glucose intolerance, impaired glucose tolerance, impaired fasting serum glucose, impaired fasting blood glucose, hyperinsulinemia, pre-diabetes, type 1 diabetes, type 2 diabetes mellitus, insulin-resistant hypertension, the metabolic syndrome, the metabolic hypertensive syndrome, (metabolic) syndrome X, the dysmetabolic syndrome, obesity, visceral obesity, and hypertriglyceridemia.

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Claim 5 (Previously presented): The method of claim 1 wherein said telmisartan, or an analog thereof, increases the activity of a PPAR subtype, PPARgamma or a PPARgamma-retinoid X receptor (PPARgamma-RXR) heterodimer.

Claim 6 (Original): The method of claim 5 wherein the activity of the PPAR subtype, PPARgamma or the PPARgamma-retinoid X receptor (PPARgamma-RXR) heterodimer is increased in combination with an increase in activity of at least one of PPARalpha and PPARdelta.

Claim 7 (Previously presented): The method of claim 5 wherein said telmisartan, or an analog thereof, is administered in a therapeutically effective amount sufficient to inhibit, slow, or delay development of at least one metabolic disorder or disease selected from the group consisting of insulin resistance, glucose intolerance, impaired glucose tolerance, impaired fasting serum glucose, impaired fasting blood glucose, hyperinsulinemia, pre-diabetes, type 1 diabetes, type 2 diabetes mellitus, insulin-resistant hypertension, the metabolic syndrome, the metabolic hypertensive syndrome, (metabolic) syndrome X, the dysmetabolic syndrome, obesity, visceral obesity, and hypertriglyceridemia.

Claim 8 (cancelled)

Claim 9 (Previously presented): The method of claim 1 wherein the telmisartan, or an analog thereof, is formulated for oral administration.

Claim 10 (Previously presented): The method of claim 1 wherein the telmisartan, or an analog thereof, is formulated for topical administration.

Claim 11 (cancelled)

Claim 12 (Previously presented): The method of claim 9 wherein the total effective daily orally administered dose is selected from the range of about 20 mg to about 1000 mg.

Claim 13 (cancelled)

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Claim 14 (Previously presented): The method of claim 9 wherein the total effective daily orally administered dose is selected from the range of about 0.05 to 100 mg/kg body weight.

Claim 15 (Original): The method of claim 1 wherein the mammal is a human child, adolescent or adult.

Claims 16-32 (cancelled)

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